TABLE 1A. Mean Values of Platelet Counts in PSL + Ceph Group*

Case number	PSL treatment ($\times 10^9$/l)	PSL + Ceph treatment ($\times 10^9$/l)	Response
1	76 ± 22	147 ± 24	+ ($P < 0.01$)
2	73 ± 30	80 ± 22	−
3	55 ± 6	94 ± 25	+ ($P < 0.01$)
4	61 ± 17	111 ± 36	+ ($P < 0.01$)
5	51 ± 9	87 ± 19	+ ($P < 0.05$)

TABLE 1B. Mean Values of Platelet Counts in Ceph-Only Group

Case number	Before treatment ($\times 10^9$/l)	Ceph treatment ($\times 10^9$/l)	Response
6	58 ± 14	57 ± 29	−
7	45 ± 12	48 ± 19	−
8	67 ± 9	62 ± 12	−
9	61 ± 12	52 ± 66	−
10	97 ± 12	100 ± 24	−

*In the PSL + Ceph group, a mean value of platelet counts under the combined treatment with PSL (at the maintenance dose) and Ceph was compared with that of platelet counts under the prior treatment with PSL (at the dose of 10 mg/day) in each case. In the Ceph-only group, a mean value of platelet counts during the Ceph treatment was compared with that of platelet counts before the treatment in each case. Each value is the mean ± SD of more than five platelets counts. Statistical differences in mean values were evaluated using the Mann-Whitney test.

tomy [1,2]. However, occasional cases are refractory to these treatments. Cepharanthin (Ceph, Kaken Shoyaku Co. Ltd., Japan), is a complex of biscoclaurine alkaloids extracted from *Stephania cepharantha* HAYATA. Ceph has been taken as an antisnake-venom drug in Japan since the 1950's [3]. Recently, Ceph has also been shown to suppress the function of the reticuloendothelial system [4] or to be involved in the proliferation/differentiation of the stem cell/megakaryocyte precursor [5].

Therefore, we applied a high-dose Ceph (10 times the usual dosage) treatment to ITP patients, and evaluated its thrombopoietic effect. Ten patients with ITP were divided into two groups and were treated with Ceph: 1) prednisolone and Ceph (PSL + Ceph), and 2) the Ceph-only group. All 5 cases in the PSL + Ceph group had histories of unsuccessful conventional PSL treatment. They were then treated with PSL combined with high-dose Ceph (60 mg/day). All 5 cases in the Ceph-only group were solely treated with Ceph (60 mg/day). In the PSL + Ceph group, 4 out of 5 patients showed a significant increase in number of platelets. In the Ceph-only group, no significant increase in number of platelets was observed. Thus, Ceph is found to be effective for increasing platelet counts when administered together with PSL (Table I). No adverse effects were observed in either group. This combined treatment of a high dose of Ceph together with PSL should be tried when various conventional internal therapies have not been particularly successful.

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Acute Hemolytic Anemia Precipitated by Myocardial Infarction and Pericardial Tamponade in G6PD Deficiency

To the Editor: Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a common metabolic disorder resulting in increased susceptibility to oxidative hemolysis. Acute hemolysis precipitated by drugs, infections, fava beans, and diabetic ketoacidosis is well-described [1]. We report on the occurrence of acute hemolytic anemia in the setting of pericardial tamponade, with post-myocardial infarction as the initial presentation of G6PD deficiency (G6PD nucleotide 1376 G→T).

A 58-year-old Chinese man was admitted to hospital with an acute anterior myocardial infarction. There was no known history of G6PD deficiency or hemolytic anemia. His only medication prior to admission was ranitidine. The patient was treated with tissue plasminogen activator, morphine, aspirin, nitroglycerin, heparin, furosemide, and propranolol. At entry the patient had a hemoglobin of 165 g/l, a white blood cell count of 13.7 × 10^9/l, and a platelet count of 359 × 10^9/l.

On the third hospital day, his blood pressure fell to 70/40, and an echocardiogram revealed pericardial tamponade. His arterial blood gases were: pH 7.46, pCO_2 27 mmHg, pO_2 64 mmHg, and bicarbonate 19 mmol/l. Serum glucose was normal. After pericardiocentesis, blood pressure and arterial blood gases normalized. Twelve hr later the patient developed hemoglobinuria, which lasted until the seventh hospital day. Hemoglobin fell to 93 g/l, and bilirubin reached 166 mmol/l by the sixth hospital day. The reticulocyte count reached 407 × 10^9/l. Numerous irregularly contracted red blood cells, blister cells, reticulocytes, and nucleated erythrocytes were present on the peripheral blood film, consistent with oxidative hemolysis (Fig. 1a). Methyl violet staining demonstrated Heinz bodies within virtually all erythrocytes (Fig. 1b). Free hemoglobin, methemalbumin, and hemopexin-heme complex were present in the serum. Serum haptoglobin was absent. There was no evidence of infection, and cultures of blood and urine were negative. Fava beans or medications known to precipitate hemolysis had not been ingested or administered.

The fluorescent spot test revealed G6PD deficiency. Hemoglobin electrophoresis, an isopropanol test, and an antibody screen were negative. G6PD activities were 0.93 and 0.30 IU/ml RBC on days 15 and 30, respectively. Amplification of the G6PD gene by polymerase chain reaction followed by sequence analysis (courtesy of Dr. E. Beutler) demonstrated a G→T mutation at nucleotide 1376 (G6PD Canton [2], Taiwan Hakka, Gifu-like, Agrigento-like [3]).

This case represents the unusual initial presentation of a common variant of G6PD deficiency. Acute hemolysis in G6PD-deficient patients has not been previously described as a complication of thrombolytic agents, myocardial infarction, or pericardial tamponade. Hyperbilirubinemia association with streptokinase administration for myocardial infarction in patients deficient in G6PD has been reported; however, in neither case was there evidence of hemolysis [4]. Although furosemide is a sulfonamide derivative, it has not been implicated as a precipitant of hemolysis and has been safely given in G6PD deficiency [5]. We propose that tissue hypoxia and metabolic acidosis from hypoperfusion resulted in an oxidative state that precipitated acute hemolysis in this case. There were no other identifiable causes for acute hemolysis in our patient.

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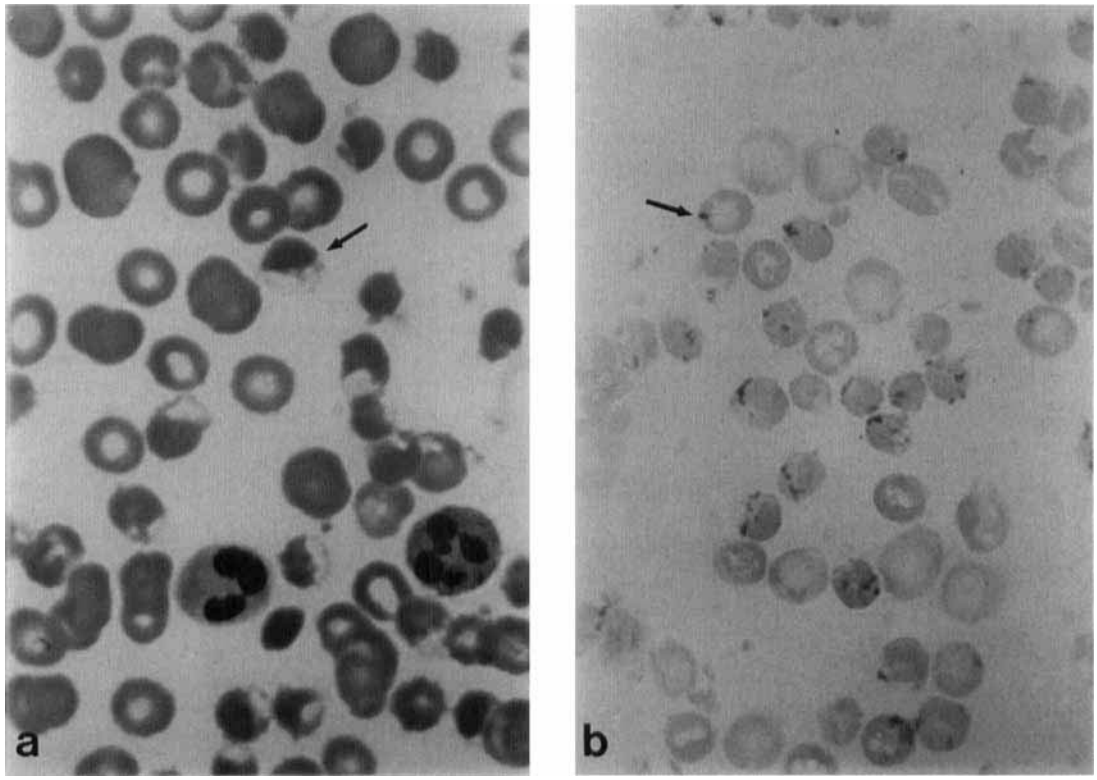

Fig. 1. a: Wright's stain: red blood cells showing numerous "blister" cells or hemighosts (arrow). b: Methyl violet stain demonstrating Heinz bodies (arrow). Wright's stain and Methyl violet stain, original magnification ×1,000.

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Burkitt's Lymphoma in a Patient With Recurrent Pericarditis

To the Editor: The rate of non-Hodgkin's lymphoma appears to be increasing, and it has been suggested that viruses appear to play a role in this recent trend [1]. Recurrent benign pericarditis is also considered to be a result of viral exposure, with subsequent activation of the immune system [2]. The patient hereby reported developed Burkitt's lymphoma following recurrent episodes of pericarditis.

A previously healthy 35-year-old Caucasian man suffered for 2 years from recurrent episodes of pericarditis. There was no evidence of exposure to various viruses, including Coxackie B and other enteroviruses, Epstein-Barr virus (EBV), cytomegalovirus (CMV), hepatitis B and C, and HIV. Common entities causing recurrent pericarditis, including lupus erythematosus and other collagen vascular disorders, tuberculosis, and rheumatic fever, were excluded; thus, a diagnosis of recurrent benign pericarditis was established.

Three months after the last event the patient presented with symptoms and signs of new-onset ascites. A small bowel series demonstrated a 30-cm portion of terminal ileum with marked irregular mucosa. Ascitic fluid aspiration revealed 49,000/mm^3 B lymphocytes showing remarkable nuclear homogeneity and the presence of vacuolated basophilic cytoplasm. The cells were immunophenotype CD19 and CD20 positive. Cytogenetic studies demonstrated t(8;14)(q24;q32) translocation, all compatible with the diagnosis of Burkitt's lymphoma. Bone marrow and meningeal involvement were not evident.

The patient received brief, intense combination chemotherapy consisting of etoposide, doxorubicin, cytosine arabinoside, cyclophosphamide, vincristin, bleomycin, and prednisone, as well as prophylactic intrathecal treatment, including methotrexate, cytosine arabinoside and hydrocortisone [3], followed by autologous bone marrow transplantation and immunotherapy. Two years later, he is in complete remission.

Recurrent pericarditis, while usually idiopathic, demonstrates the presence of IgM antibodies to enteroviruses, especially Coxackie B in more than one-half of cases, and about one-third of patients have antimyocardial antibodies [2].

The pathogenesis of Burkitt's lymphoma is thought to involve chronic antigenic stimulation caused by malaria, EBV, or HIV infection initiating polyclonal B-cell proliferation, from which the neoplastic clone could emerge upon accumulation of multiple genetic events involving activation